Letters to the Editor

High alcohol intake and slow progression to AIDS

It is not yet understood why some HIV-1 infected persons develop AIDS within a few years after infection and others remain healthy and with a normal CD4 cell count for many years. In order to assess the influence of different epidemiological and behavioural factors and the role of infection with pathogens which might act as co-factors for disease progression in HIV-1 infected people, a multicentre study was started in Madrid in September 1992.

Rapid progressors (RP) and slow progressors (SP) were checked in a group of 1783 HIV-infected persons regularly attending three medical centres in Madrid. Definition criteria were for RP: infection occurred in the last 5 years and with a current CD4 + count repeatedly below 200/mm;[3] and for SP: more than 8 years of confirmed HIV-1 infection and with the number of CD4 + cells consistently above 500/mm,[3] in the absence of any antiretroviral therapy and without symptoms. One hundred persons (5·6%) met the criteria for SP and 12 (0·7%) for RP. Of 48 SP with a CD4 count monitored for more than 5 years, 16 (33%) were absolute non-progressors, maintaining a normal and stable CD4 + count.

Variables more frequently recognised in the SP group compared with the RP group were: previous injecting drug addiction (IDA) practices ($p = 0·0002$), low cultural level ($p = 0·0023$), younger age beginning high-risk practices ($p = 0·0039$), male gender ($p = 0·0370$) and prolonged high alcohol intake ($p = 0·0391$), defined as consumption of alcohol above 100 g daily for more than 3 years. Co-infection with hepatitis B and C viruses or other infectious agents which could act as co-factors was not seen more frequently in RP. Categorising by the route of infection (sexually or parenterally), a younger age beginning high-risk practices was associated with SP in injecting drug users, and female gender was associated with RP in people infected through sexual contact. Chronic high alcohol intake showed a strong association with SP amongst injected drug users although it did not achieve significance ($p = 0·0995$). There was no evidence linking this effect to any particular drink or liquor. Nevertheless, in three HIV-positive heavy drinkers with SP, an enhancement of plasma HIV-RNA was not seen two weeks after stopping the intake of alcohol, or a fall after resuming alcohol consumption.

Although there have been reports of rapid progression to AIDS in alcoholic HIV-infected patients,[1] longitudinal studies in large cohorts have not been able to find any association between high alcohol intake and worse prognosis in HIV-infected patients.[2 3] We postulate that in HIV-infected subjects with preserved immune status, chronic high alcohol intake could have a protective effect against CD4 + depletion, as has recently been proposed for two other immunosuppressive substances, as corticosteroids[4] and cyclosporin A.[5] Two main reasons could explain this unexpected beneficial effect of alcohol. First, some substances present in many alcoholic drinks, as flavonoids in red wine, have a powerful antioxidant activity,[6] which can reduce virus expression in infected cells. Second, ethanol can suppress the activation of lymphocytes and monocytes/macrophages, which function as an important reservoir for the virus. Indirectly, expression of the virus in infected cells could be suppressed, causing a decline in HIV viraemia and perhaps yielding a prolonged survival in these patients. Of course, before recommending a couple of whiskies daily, doctors and patients should be aware that this hypothetical benefit of alcohol on HIV replication needs to be balanced with other disadvantageous effects of alcoholism, mainly on liver function and nutritional status, which by other means might cause immune dysfunction.

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Dietary intervention in HIV: a comparison of patients receiving oral, enteral and parenteral nutrition

Weight loss is an important complication of human immunodeficiency virus (HIV) infection. Nutritional deficiency contributes to the progression of disease and susceptibility to opportunistic infections and weight loss is a major factor associated with the time of death.[1–3] Weight loss associated with HIV is a complex process and may result from decreased nutrient intake, impaired nutrient absorption or increased nutritional requirements. The optimum method of delivering nutritional support at each stage of infection has not been established. We therefore reviewed patients referred to a designated HIV dietician over a two year period and compared the characteristics and outcome of patients receiving dietary advice only, oral supplementation, enteral feeding and parenteral nutrition.

Information on all adult patients seen by the HIV dietician over a two year period during 1993–95 was obtained from the medical and

Comparison of oral, enteral and parenteral feeding in HIV

	Advice only	*Oral supplements*	*Enteral nutrition*	*Parenteral nutrition*
No	14	24	10	3
No with AIDS (%)	3 (27%)	7 (29%)	9* (90%)	3 (100%)
Mean CD4 count	177	130	14†	10
Range (cells/mm³)	(10–560)	(10–520)	(0–30)	(0–20)
Mean BMI	22·6‡	20·2	20·2	—
(range)	(20–25)	(17–23)	(19–23)	
Mean weight (kg)	64·1	62·9	60·5	50·5
(range)	(41·3–92·1)	(45–76)	(49·4–70·8)	(47·5–52)
Mean wt loss (kg)	1·0	9·5§	18‖	22·2
(range)	(0–9·0)	(0–36)	(5–38)	(16·5–28)
(mean % wt loss)	(1·5%)	(13%)	(13%)	(31%)
Mean wt change (kg)	−0·6	0·36	3·01	—
over three months (range)	(−7·0 to +9·0)	(−11·0 to +14·5)	(−4·1 to +8·8)	—

*p < 0·01 comparing proportion of patients with AIDS receiving enteral nutrition with group receiving advice only.
†p < 0·025 comparing mean CD4 cell count in patients receiving enteral nutrition with group receiving advice only.
‡p < 0·01 comparing mean BMI in patients receiving advice only with groups receiving oral or enteral nutrition.
§p <0·005 comparing mean prior weight loss in patients receiving oral supplements with group receiving advice only.
‖ p < 0·0005 comparing mean prior weight loss in patients receiving enteral nutrition with groups receiving advice or oral supplements.

dietary records. Dietary interventions were based on the assessment of the dietician and the preferences of the patients involved. Proportions of patients with the acquired immune deficiency syndrome (AIDS) were compared by chi square analysis. CD4 counts, basal metabolic index (BMI) and weights of patients in each group were compared by unpaired *t* test analysis.

Fifty one patients were seen by the designated dietician during the period of the study. Of these 48 (94%) were male and their mean age was 38·5 years (range 18–57 years). Forty two patients (82%) were Caucasian and nine (18%) were of Asian origin. Thirty four patients (67%) had a diagnosis of AIDS.

Fourteen patients (27%) received dietary advice only. This group had a mean CD4 + cell count of 177/mm³; mean BMI was 22·6 and mean weight loss prior to dietary consultation was only 1 kg. The mean weight change over the three month period following dietary advice was a reduction of 0·6 kg. Twenty four patients (47%) were given oral nutritional supplements. This group of patients had more advanced disease with a mean CD4 + cell count of 130/mm³; mean BMI was 20·2 and mean weight loss prior to dietary consultation was 9·5 kg. Oral supplementation was associated with a mean weight gain of 0·36 kg over three months. Ten patients (20%) received enteral nutrition which was administered via a percutaneous endoscopic gastrostomy (PEG) tube in two cases. The mean CD4 + cell count of patients receiving enteral nutrition was only 14/mm³ and 90% had a diagnosis of AIDS. Mean BMI was 20·2 and mean weight loss prior to dietary consultation was 18 kg representing a 23% reduction of baseline weight. Enteral feeding was associated with a weight gain of over 3 kg over three months. Three patients received parenteral feeding, all with a diagnosis of AIDS complicated by enteropathy and opportunistic infections. Long-term nutrition was self-administered via a Hickman line (data summarised in the table).

The patients in this study had relatively advanced disease and the intensity of dietary intervention showed a significant correlation with the stage of disease and previous weight loss. However, the results show that nutritional support, particularly enteral feeding, was frequently delayed until very substantial weight loss had occurred. More aggressive dietary intervention should therefore be considered, particularly in patients with advanced disease and multiple concurrent problems.

Dietary advice and oral nutritional supplementation are useful in the early stages of infection and may help to maintain baseline weight.[4] Our results showed that oral supplementation and enteral nutrition were both associated with a mean weight gain over three months. Enteral feeding produces an increase in body cell mass[5] and the magnitude of the response to enteral feeding in our patients was similar to that reported previously. Parenteral nutrition can be effective but is very expensive and should be reserved for selected patients who are unable to tolerate enteral feeding.

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The "Kumasi modified" two glass urine test and urinary schistosomiasis

The aetiology of non gonococcal urethritis is myriad[1] and with the exception of chlamydia infection, investigations are often unrewarding. In the assessment of a patient with suspected urethritis but no overt discharge, the two glass urine test[2] is often carried out. We have modified this test by centrifuging urine